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                  Substances (PICCS) has been added to CHEMLIST
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=> s lung cancer cell line and diagnos?

L1 150 LUNG CANCER CELL LINE AND DIAGNOS?

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L2 129 L1

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L3 92 DUPLICATE REMOVE L2 (37 DUPLICATES REMOVED)

=> d 1-10 bib ab

L3 ANSWER 1 OF 92 CAPLUS COPYRIGHT 2000 ACS
AN 1998:801200 CAPLUS
DN 130:151827
TI MUC1 is a novel marker for the type II pneumocyte lineage during lung carcinogenesis
AU Jarrard, Julia A.; Linnoila, R. Ilona; Lee, HyeRan; Steinberg, Seth M.; Witschi, Hanspeter; Szabo, Eva
CS Cell and Cancer Biology Department, Medicine Branch, Division of Clinical Sciences, National Cancer Institute, Rockville, MD, 20850, USA
SO Cancer Res. (1998), 58(23), 5582-5589
CODEN: CNREA8; ISSN: 0008-5472
PB AACR Subscription Office
DT Journal
LA English
AB Abnormalities in mucin-type glycoprotein expression have been documented in a variety of cancers, identifying these mols. as targets for immunol. based therapies and prognostic/**diagnostic** assays. The authors examd. the expression of the membrane-bound MUC1 mucin in normal, histol. atypical, and neoplastic lung to det. its potential contribution to lung carcinogenesis. In vivo, intense MUC1 immunoreactivity was present in normal type II pneumocytes as well as in a range of atypical lesions derived from type II cells and >60% of primary and metastatic non-small cell lung cancers. Expression was not assocd. with altered survival, although it was highly correlated with the adenocarcinoma histol. A carcinogenesis model using 4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone-exposed hamsters revealed that MUC1 mRNA increased prior to the histol. appearance of tumors. In vitro studies using MUC1 expressing non-small cell **lung cancer** cell **lines** revealed that differentiation away from a type II cell lineage was assocd. with dramatic down-regulation of MUC1. The authors propose that MUC1 is a powerful new marker for the type II pneumocyte cell lineage that allows us to follow the type II pneumocyte lineage during the

process of lung carcinogenesis.
RE.CNT 35
RE
(1) Abe, M; Cancer Res 1989, V49, P2834 CAPLUS
(3) Agrawal, B; Nat Med 1998, V4, P43 CAPLUS
(6) Chambers, J; J Cell Sci 1994, V107, P413 CAPLUS
(7) Chen, L; J Clin Invest 1995, V96, P2775 CAPLUS
(8) Dahiya, R; Biochem Mol Biol Int 1995, V35, P351 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 92 MEDLINE DUPLICATE 1
AN 1998338122 MEDLINE
DN 98338122
TI Molecular characterization of the human delta opioid receptor in lung cancer.
AU Schreiber G; Campa M J; Prabhakar S; O'Briant K; Bepler G; Patz E F Jr
CS Department of Radiology, Duke University Medical Center, Durham, North Carolina 27710, USA.. schre002@mc.duke.edu
SO ANTICANCER RESEARCH, (1998 May-Jun) 18 (3A) 1787-92.
Journal code: 59L. ISSN: 0250-7005.
CY Greece
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199810
AB A variety of neuropeptide receptors have been detected in human lung cancer. They are thought to play a role in autocrine/paracrine regulation of cell growth, and may be clinically useful as **diagnostic**, prognostic or therapeutic targets. The current study characterizes the molecular structure of the delta opioid receptor and its gene expression level in **lung cancer cell lines** relative to normal human lung using a sensitive RT-PCR approach. The goals of this investigation were a) to define the correlation between receptor binding and gene expression in **lung cancer cell lines**, and b) to determine the cDNA sequence integrity of this receptor in comparison to the receptor recently found in human brain. Five small cell lung cancer (SCLC) cell lines revealed size-matched RT-PCR products which strongly hybridized to the human brain delta opioid receptor probe. One of three non-small cell lung cancer (NSCLC) cell lines (NCI-H23), known to be negative by binding analysis, demonstrated low level expression. No gene expression was found in normal human lung. RT-PCR products from two SCLC cell lines (SCLC-22H and 16HC) as well as the low level expressing NSCLC cell line (NCI-23) were subjected to bidirectional DNA sequence analysis and the receptor ends were resolved using a 3'-end RACE and 5'-end gene-specific approach. The isolated cDNA sequences proved to be identical to the published human brain delta opioid receptor sequence. These data show that lung cancers with neuroendocrine features express human brain delta opioid receptors in contrast to normal lung, and that the delta opioid receptor mRNA in lung cancer is not mutated. This unique feature of lung cancer may be exploitable for **diagnostic**, prognostic, and therapeutic strategies.

L3 ANSWER 3 OF 92 MEDLINE DUPLICATE 2
AN 1998132666 MEDLINE
DN 98132666
TI Antisense inhibition of the PTI-1 oncogene reverses cancer phenotypes.

AU Su Z; Goldstein N I; Fisher P B
CS Departments of Pathology and Urology, Herbert Irving Comprehensive Cancer Center, Columbia University College of Physicians and Surgeons, New York, NY 10032, USA.
NC CA35675 (NCI)
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1998 Feb 17) 95 (4) 1764-9.
Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199805
AB The genetic alterations and molecular events mediating human prostate cancer development and progression remain to be defined. Rapid expression cloning and differential RNA display detect a putative oncogene, prostate tumor-inducing gene 1 (PTI-1), that is differentially expressed in human prostate (as well as breast, colon, and small cell **lung**) **cancer cell lines**, patient-derived prostate carcinomas, and blood from patients with metastatic prostate cancer. PTI-1 consists of a unique 5' untranslated region (5' UTR) with significant sequence homology to Mycoplasma hyopneumoniae 23S ribosomal RNA juxtaposed to a sequence that encodes a truncated and mutated human elongation factor 1alpha (Trun-EF). Stable expression of a nearly full-length 1.9-kb PTI-1 gene, but not the separate PTI-1 5' UTR or Trun-EF region, in normal rat embryo fibroblast cells, CREF-Trans 6, induces an aggressive tumorigenic phenotype in athymic nude mice. Blocking PTI-1 expression with antisense PTI-1 results in reversion of transformed PTI-1-expressing cells to a more normal cellular morphology with suppression in both anchorage-independent growth and tumorigenic potential in athymic nude mice. These findings document that PTI-1 is indeed an oncogene, and directly blocking PTI-1 expression can nullify cancer phenotypes. In these contexts, PTI-1 not only represents a gene with discriminating **diagnostic** properties but also may serve as a target for the gene-based therapy of human prostate and other cancers.

L3 ANSWER 4 OF 92 MEDLINE
AN 1998239450 MEDLINE
DN 98239450
TI Predicting chemotherapeutic response to small-cell lung cancer of platinum compounds by thallium-201 single-photon emission computerized tomography.
AU Tokuchi Y; Isobe H; Takekawa H; Hanada T; Ishida T; Ogura S; Itoh K; Furudate M; Saito K; Kawakami Y
CS First Department of Medicine, School of Medicine, Hokkaido University, Nishi, Sapporo, Japan.
SO BRITISH JOURNAL OF CANCER, (1998 Apr) 77 (8) 1363-8.
Journal code: AV4. ISSN: 0007-0920.
CY SCOTLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199807
EW 19980705
AB Thallium-201 single-photon emission computerized tomography (SPECT) was

used to clarify the relationship between 201Tl uptake and the response in chemotherapy to platinum compounds in 21 patients with small-cell lung cancer. 201Tl-SPECT scans were obtained twice: at 15 min (early scan) and 120 min (delayed scan) after an intravenous injection of 111 MBq (3 mCi) of thallium-201 chloride. We obtained the uptake ratio from each scan and calculated the retention index:uptake ratio = region of interest uptake/contralateral normal lung uptake; retention index = (delayed ratio - early ratio)/early ratio. After 201Tl scintigraphy, 12 patients received chemotherapy consisting of platinum compounds and nine were treated with chemoradiation. Among patients receiving only chemotherapy, the retention index correlated with the responses to chemotherapy. In an in vitro study, ouabain, an inhibitor of the Na,K-ATPase pump, reduced sensitivity to cisplatin and inhibited intracellular thallium uptake in the small-cell **lung cancer cell line**. These studies suggest that 201Tl-SPECT is a useful indicator of response to chemotherapy with platinum compounds in small-cell lung cancer, and that Na,K-ATPase is commonly involved in transporting both thallium and platinum compounds into cancer cells.

L3 ANSWER 5 OF 92 MEDLINE
AN 1998437166 MEDLINE
DN 98437166
TI Proliferation of human non-small-cell **lung cancer cell lines**: role of interleukin-6.
AU Bihl M; Tamm M; Nauck M; Wieland H; Perruchoud A P; Roth M
CS Division of Pneumology, Department of Internal Medicine and Research, University Hospital, Basel, Switzerland.
SO AMERICAN JOURNAL OF RESPIRATORY CELL AND MOLECULAR BIOLOGY, (1998 Oct) 19 (4) 606-12.
Journal code: AOB. ISSN: 1044-1549.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199901
EW 19990104
AB Interleukin-6 (IL-6) is involved in regulation of the immune response, acute phase reaction, and cell proliferation. The aim of this study was to investigate whether IL-6 is implicated in cell proliferation of human non-small-cell lung cancer (NSCLC) cell lines. We analyzed IL-6 messenger RNA (mRNA) and protein expression in eight NSCLC cell lines: A549, Calu3, Calu6, H23, H522, H810, H1155, and H1299. The A549, Calu3, Calu6, and H23 cell lines expressed IL-6 mRNA and protein. In these cell lines, fetal calf serum (FCS) significantly increased cell proliferation as assessed by thymidine incorporation. In the presence of IL-6 antisense oligonucleotides, both proliferation and IL-6 synthesis were downregulated. In contrast, IL-6 mRNA and protein could not be detected in the NSCLC cell lines H522, H810, H1155, and H1299. In these NSCLC cell lines, FCS only marginally increased cell proliferation and IL-6 antisense oligonucleotides did not affect cell proliferation. The addition of neither exogenous IL-6 nor neutralizing anti-IL-6 antibodies affected cell

proliferation in any of the experiments. Our data thus provide evidence that intracellular IL-6 is required in the control of cell proliferation in a subset of human NSCLC cell lines. We suggest the existence of two subtypes of NSCLC, an IL-6-dependent and an IL-6-independent type.

L3 ANSWER 6 OF 92 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 3
AN 1998:138216 BIOSIS
DN PREV199800138216
TI Distribution profile of urinary human chorionic gonadotropin subunits: Assay development for lung and breast cancer.
AU De Castro-Bernas, G. (1); Garcia, I. J.
CS (1) Res. Cent. Natural Sciences, Espana, Manila 1008 Philippines
SO Medical Science Research, (Jan., 1998) Vol. 26, No. 1, pp. 21-23. ISSN: 0269-8951.
DT Article
LA English
AB The alpha and beta subunits of human chorionic gonadotropin (hCG) in 117 urine samples from patients clinically **diagnosed** to have lung cancer, in 50 urine samples from breast cancer patients, and 70 urine samples from nonpregnant normal and pregnant subjects, were separated using ion-exchange chromatography. Determination of the autocrine hormone in the samples from non-small cell lung carcinoma cases revealed that 69% had significantly elevated alpha subunits ($p < 0.05$). Of the 19 small cell lung cancer cases screened, 26% showed elevated alpha peaks. Of the 50 breast cancer cases, 64% had significantly elevated alpha peaks ($p < 0.05$). In non-pregnant urine samples, 66% and 90% respectively showed elevated beta peaks. For comparison, we also investigated the distribution profile of hCG subunits in established **lung cancer cell lines**. There was a marked increase in alpha subunit in the spent medium (supernatant) from SL-6 (large cell carcinoma). A moderate rise in the alpha subunit occurred in both A-549 (lung adenocarcinoma) and SLNI-52 (tracheo-bronchial carcinoma). Supernatant from primary culture of mammary cancer explant also showed an elevated alpha subunit.

L3 ANSWER 7 OF 92 CAPLUS COPYRIGHT 2000 ACS
AN 1997:341932 CAPLUS
DN 126:315953
TI Heterogeneous ribonucleoprotein and DNA thereof for use in early cancer detection
IN Mulshine, James L.; Tockman, Melvyn S.
PA United States Dept. of Health and Human Services, USA; John Hopkin's University
SO PCT Int. Appl., 171 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9712975	A1	19970410	WO 1996-US15825	19961002

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA

US 5994062 A 19991130 US 1995-538711 19951002
AU 9673864 A1 19970428 AU 1996-73864 19961002
EP 861323 A1 19980902 EP 1996-936141 19961002
R: CH, DE, FR, GB, IT, LI
JP 2000500322 T2 20000118 JP 1997-514401 19961002
WO 9814469 A2 19980409 WO 1997-US17714 19971002
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
AU 9746635 A1 19980424 AU 1997-46635 19971002
PRAI US 1995-538711 19951002
US 1996-725027 19961002
WO 1996-US15825 19961002
WO 1997-US17714 19971002

AB The present invention is a purified and isolated epithelial protein, peptide and variants thereof whose increased presence in an epithelial cell is indicative of precancer. One epithelial protein which is an early detection marked for lung cancer was purified from two human **lung cancer cell lines**, NCI-H720 and NCI-H157. Using a six-step procedure, the epithelial protein was purified using a Western blot detection system under both non-reducing and reducing conditions. Purifn. steps included anion exchange chromatog., preparative isoelec. focusing, polymer-based C18HPLC and analytic C4HPLC. After an approx. 25,000 fold purifn. the immunostaining protein was >90% pure as judged by Coomassie Blue staining after reducing SDS-PAGE. The primary epithelial protein shares sequence homol. with the heterogeneous nuclear ribonucleoprotein (hnRNP) A2. A minor co-purifying epithelial protein shares sequence homol. with the splice variant hnRNP-B1. Mol. anal. of primary normal bronchial epithelial cell cultures demonstrated a low level the epithelial protein expression, consistent with immunohistochem. staining of clin. samples, and an increased level of expression in most lung cancer cells. The epithelial protein is a marker of epithelial transformation in lung, breast, bone, ovary, prostate, kidney, melanoma and myeloma and may be causal in the process of carcinogenesis. Methods are provided for monitoring the expression of the epithelial protein, peptides and variants using mol. and immunol. techniques as a screen for precancer and cancer in mammals. A method of computerized **diagnoses** of cancer and precancer is provided which detects levels of hnRNP mRNA.

L3 ANSWER 8 OF 92 CAPLUS COPYRIGHT 2000 ACS
AN 1997:492171 CAPLUS
DN 127:107995
TI A humanized monoclonal antibody to a cell line derived from a human small cell lung carcinoma
IN Bendig, Mary; Saldana, Jose; Jones, Tarran
PA Merck Patent Gmbh, Germany
SO Eur. Pat. Appl., 91 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 781847	A1	19970702	EP 1996-117154	19961025

R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE

PRAI EP 1995-117407 19951106

AB A new humanized and reshaped monoclonal antibody derived from murine monoclonal antibody 15 (MAb 15) raised against the small cell **lung cancer cell line** TKB-2 is described for **diagnostic** and therapeutic use. Genes for the light and heavy chains of this antibody are also described.

L3 ANSWER 9 OF 92 BIOSIS COPYRIGHT 2000 BIOSIS

AN 1997:233982 BIOSIS

DN PREV199799533185

TI Comparison of c-myc expression in normal human bronchial epithelial (NHBE) cells and **lung cancer cell lines** using quantitative fluorescence-based reverse transcriptase/polymerase chain reaction (RT/PCR.

AU Fields, W. R. (1); Bombick, D. W.; Atkins, S. S.; Doolittle, D. J.

CS (1) Integrated Toxicol. Program, Duke Univ., Durham, NC 27710 USA

SO Proceedings of the American Association for Cancer Research Annual Meeting, (1997) Vol. 38, No. 0, pp. 564.
Meeting Info.: Eighty-eighth Annual Meeting of the American Association for Cancer Research San Diego, California, USA April 12-16, 1997
ISSN: 0197-016X.

DT Conference; Abstract

LA English

L3 ANSWER 10 OF 92 MEDLINE DUPLICATE 4

AN 97287103 MEDLINE

DN 97287103

TI Granulocyte colony-stimulating factor and interleukin-6-producing **lung cancer cell line**, LCAM.

AU Inoue M; Minami M; Fujii Y; Matsuda H; Shirakura R; Kido T

CS Division of Organ Transplantation, Osaka University Medical School, Japan.

SO JOURNAL OF SURGICAL ONCOLOGY, (1997 Apr) 64 (4) 347-50.
Journal code: K79. ISSN: 0022-4790.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Cancer Journals

EM 199707

EW 19970704

AB BACKGROUND AND OBJECTIVES: We describe a case of granulocyte colony-stimulating factor (GCSF) and interleukin-6 (IL-6)-producing lung cancer. METHODS: A 53-year-old man underwent left upper lobectomy under **diagnosis** of lung cancer. The tumor obtained by a preoperative biopsy was analyzed. RESULTS: Preoperative data showed leukocytosis with left-shift of leukocytic morphology and thrombocytosis and an elevated serum GCSF level. Histological examination revealed poorly differentiated adenocarcinoma. A cell line, named LCAM, was established from the tumor and the cytokines in the culture medium were measured by enzyme immunoassay. GCSF and IL-6 were produced in large amounts by LCAM, but granulocyte-macrophage colony-stimulating factor (GMCSF) and interleukin-3 (IL-3) were not. A proportion of LCAM expressed GCSF receptor on the cell surface, but IL-6 receptor could not be detected. LCAM proliferation was

inhibited in the culture with antihuman GCSF antibody in a dose-dependent manner. CONCLUSIONS: We suggest that LCAM proliferation is positively regulated by GCSF.

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=> s breast cancer cell lines and diagnos?

L4          130 BREAST CANCER CELL LINES AND DIAGNOS?

=> s l4 range=,1998
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SEARCH ENDED BY USER
L5           74 L4

=> s breast cancer cell line and diagnos?

L6          257 BREAST CANCER CELL LINE AND DIAGNOS?

=> s l6 range=,1998

L7          185 L6

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DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS, CAPLUS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n

PROCESSING COMPLETED FOR L7
L8           141 DUPLICATE REMOVE L7 (44 DUPLICATES REMOVED)

=> d 1-10 bib ab


L8   ANSWER 1 OF 141  CAPLUS  COPYRIGHT 2000 ACS
AN   1998:709189  CAPLUS
DN   129:327727
TI   Mammalian ubiquitin isopeptidases that control cell proliferation and
     their cDNAs and diagnosis and treatment of cancer
IN   Naviglio, Silvio; Soncini, Chiara; Capra, Maria; Goubin, Francoise;
     Matoskova, Bronia; Di Fiore, Pier Paolo; Draetta, Giulio Francesco;
     Bosari, Silvano
PA   Istituto Europeo Di Oncologia S.r.l., Italy
SO   PCT Int. Appl., 75 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ---------------   ----  --------      ---------------  --------
PI   WO 9848020         A2   19981029      WO 1998-IT84     19980410
     WO 9848020         A3   19990121
         W:  AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
             DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG,
             KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX,
             NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT,
             UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,
             FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI,
             CM, GA, GN, ML, MR, NE, SN, TD, TG
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AU 9870779 A1 19981113 AU 1998-70779 19980410
PRAI IT 1997-MI905 19970418
WO 1998-IT84 19980410
AB The present invention relates to ubiquitin isopeptidases (UBPs), to the polynucleotides encoding them, to methods for detecting such proteins and regulating their cellular activities. Three human UBP cDNAs were cloned and sequenced. The deubiquitinating activity of one of the UBPs was demonstrated using, as substrate, cell exts. from cells treated with proteasome inhibitors. This UBP was found to play a crit. role in cell proliferation: cells microinjected with antisense constructs failed to enter S phase. Levels of this UBP were downregulated in contact-inhibited cells while in non-contact-inhibited osteosarcoma cells no downregulation was obsd. In human **breast cancer cell line** MCF7, the UBP levels were significantly higher than in normal tissue.

L8 ANSWER 2 OF 141 CAPLUS COPYRIGHT 2000 ACS
AN 1998:709183 CAPLUS
DN 129:329393
TI A novel mucin variant as a marker for epidermal carcinomas and a cDNA encoding it
IN Schwaeble, Wilhelm
PA University of Leicester, UK
SO PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9848014	A1	19981029	WO 1998-GB1184	19980423

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9870677 A1 19981113 AU 1998-70677 19980423
PRAI GB 1997-8190 19970423
WO 1998-GB1184 19980423
AB A mucin variant (MUC-B1) that can be used as a marker for breast and ovarian cancers and epithelial cancers in general is described. A cDNA encoding the protein is also described. As a marker, it shows greater specificity than previously known mucin markers. Antibodies are prepd. for immunochem. detection of the protein. The marker was first identified as the antigen recognized by a monoclonal antibody specific to a **breast cancer cell line**. The MUC-B1 variant was absent from normal breast epithelium but was found in 60% of breast cancer tissue samples. A cDNA was cloned by screening cDNA expression libraries with the antibody.

L8 ANSWER 3 OF 141 CAPLUS COPYRIGHT 2000 ACS
AN 1998:672565 CAPLUS
DN 129:286741
TI Dil2 gene and methods and compositions for **diagnosis** and treatment of breast cancer

IN Vournakis, John N.; Seth, Arun K.; Papas, Takis S.
PA MUSC Foundation for Research Development, USA
SO PCT Int. Appl., 106 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9842725	A1	19981001	WO 1998-US5629	19980320
W: AL, AM, AU, AZ, BA, BB, BG, BR, BY, CA, CN, CU, CZ, EE, GE, GH, GW, HU, ID, IL, IS, JP, KG, KP, KR, KZ, LC, LK, LR, LT, LV, MD, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9867685	A1	19981020	AU 1998-67685	19980320
EP 1007535	A1	20000614	EP 1998-913038	19980320
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRAI US 1997-44425 19970321
WO 1998-US5629 19980320

AB The present invention relates to a novel gene, Di12, that is differentially expressed as a 1.35 kb RNA in breast cancer tissues and cell lines, and in several normal tissues. The full length cDNA encodes a protein of 339 amino acids. Antibodies to the gene product were developed to investigate the expression of Di12 in **breast cancer cell-lines** and tumors. The Di12 protein was found in tissue sections of infiltrating ductal carcinomas (IDCs), but not in benign or normal breast specimens. Di12 was also present in IDC-breast cancer patient sera, and its expression level increased markedly if IDC was accompanied by lymph node or distal metastases. As IDC constitutes .apprx.70 % of breast cancers seen clin., the level of Di12 expression is useful for diseases **diagnosis** predicting disease progression and monitoring a therapeutic treatment.

L8 ANSWER 4 OF 141 CAPLUS COPYRIGHT 2000 ACS
AN 1998:493691 CAPLUS
DN 129:121167
TI CSK homologous kinase (CHK) as a marker for breast cancer in the **diagnosis** and treatment of the disease
IN Avraham, Hava; Groopman, Jerome E.
PA Beth Israel Deaconess Medical Center, USA; Avraham, Hava; Groopman, Jerome E.
SO PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9830704	A1	19980716	WO 1998-US420	19980107
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5981201	A	19991109	US 1997-876882	19970616

AU 9858199 A1 19980803 AU 1998-58199 19980107
EP 972050 A1 20000119 EP 1998-901751 19980107
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
PRAI US 1997-35228 19970108
US 1997-876882 19970616
WO 1998-US420 19980107
AB CSK homologous kinase (CHK) is shown to be present in tumorous breast tissue but absent from adjacent normal tissue. Accordingly, the enzyme can be a marker for detecting and monitoring the disease and a target for its treatment. The kinase is shown to interact with the erbB2 protein via the SH2 domain in the T47D **breast cancer cell line** upon stimulation of cells with heregulin. Interaction between CHK and erbB2 could be inhibited by the phosphotyrosine-contg. peptides derived from the receptor.

L8 ANSWER 5 OF 141 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:498809 BIOSIS
DN PREV199800498809
TI Apoptosis induction and potent antiestrogen receptor-negative breast cancer activity in vivo by a retinoid antagonist.
AU Fanjul, Andrea N.; Piedrafita, F. Javier; Al-Shamma, H.; Pfahl, Magnus (1)
CS (1) MAXIA Pharm. Inc., 10835 Altman Row, Suite 250, San Diego, CA 92121 USA
SO Cancer Research, (Oct. 15, 1998) Vol. 58, No. 20, pp. 4607-4610. ISSN: 0008-5472.
DT Article
LA English
AB Close to 180,000 women will be **diagnosed** with breast cancer this year in the United States and more than 43,000 will die from this disease. Antiestrogens have shown promise, but they can only be effective against estrogen-dependent stages of the disease. We identify here a retinoid antagonist, MX781, that is effective against estrogen receptor-positive and -negative breast cancer cells. Although classical retinoids show limited efficacy and significant side effects, this novel compound kills breast cancer cells by inducing apoptosis and is effective against estrogen receptor-negative human breast cancer tumors in vivo. Remarkably, MX781 is well tolerated and does not seem to have significant toxicity. This novel retinoid antagonist, therefore, represents a promising new candidate for the treatment of breast cancer.

L8 ANSWER 6 OF 141 MEDLINE DUPLICATE 1
AN 1998368411 MEDLINE
DN 98368411
TI Limitations of specific reverse-transcriptase polymerase chain reaction markers in the detection of metastases in the lymph nodes and blood of breast cancer patients.
AU Bostick P J; Chatterjee S; Chi D D; Huynh K T; Giuliano A E; Cote R; Hoon D S
CS Department of Molecular Oncology, John Wayne Cancer Institute at Saint John's Health Center, Santa Monica, CA 90404, USA.
SO JOURNAL OF CLINICAL ONCOLOGY, (1998 Aug) 16 (8) 2632-40. Journal code: JCO. ISSN: 0732-183X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals; Cancer Journals
EM 199811
EW 19981101
AB PURPOSE: This study was performed to evaluate the potential of specific mRNA markers to detect micrometastases by reverse-transcriptase polymerase chain reaction (RT-PCR) and Southern blot analysis of sentinel lymph nodes (SNs) and blood from patients with breast cancer. PATIENTS AND METHODS: We assessed the specificity of carcinoembryonic antigen (CEA), cytokeratin-19 (CK-19), CK-20, gastrointestinal tumor-associated antigen-733.2 (GA733.2), and mucin-1 (MUC-1) in the blood of healthy donors (n = 13) and lymph nodes from patients without cancer (n = 3) by RT-PCR assay. The sensitivity of the RT-PCR assay for the target mRNA markers was assessed in **breast cancer cell lines** (n = 4), primary breast tumors (n = 8), and the frozen sections of SNs (n = 22) from 22 patients with American Joint Committee on Cancer (AJCC) stages I to IIIA breast cancer. RESULTS: CK-20 was the only mRNA marker not detected in lymph nodes or blood from patients without cancer. Both the blood and lymph nodes from patients without cancer expressed CEA, CK-19, GA733.2, and MUC-1 mRNA. All four **breast cancer cell lines** and six of eight primary breast tumors expressed all five mRNA markers. Expression of mRNA by the RT-PCR assay in the frozen-section SNs (n = 12) without metastases by conventional histopathology ranged from 8% (CK-20) to 92% (GA733.2). Detection of RT-PCR cDNA products in frozen-section SNs was increased with Southern blot analysis compared with ethidium bromide gel electrophoresis (EtBr) for all mRNA markers except CK-19. CONCLUSION: CEA, CK-19, GA733.2, and MUC-1 show no **diagnostic** value as mRNA markers for the detection of micrometastases by the RT-PCR assay because they are expressed in the blood and lymph nodes of patients without cancer. Further studies are needed to assess the sensitivity of CK-20 to detect micrometastases by the RT-PCR assay in the blood and frozen-section SNs of patients with breast cancer.

L8 ANSWER 7 OF 141 MEDLINE DUPLICATE 2
AN 1998418914 MEDLINE
DN 98418914
TI Immunocytochemical detection of somatostatin receptors sst1, sst2A, sst2B, and sst3 in paraffin-embedded breast cancer tissue using subtype-specific antibodies.
AU Schulz S; Schulz S; Schmitt J; Wiborny D; Schmidt H; Olbricht S; Weise W; Roessner A; Gramsch C; Holltt V
CS Department of Obstetrics and Gynecology, Otto-von Guericke-University, Magdeburg, Germany.
SO CLINICAL CANCER RESEARCH, (1998 Sep) 4 (9) 2047-52.
Journal code: C2H. ISSN: 1078-0432.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199901
EW 19990104

AB The long-acting somatostatin analogue octreotide (SMS 201-995) inhibits growth of certain **breast cancer cell lines** in vivo and in vitro. Because the antiproliferative action of octreotide depends on at least the presence of somatostatin receptors, it is crucial to determine the pattern of somatostatin receptor protein expression on the tumor cells. In the present study, we have raised polyclonal antibodies to somatostatin receptor subtypes (ssts) sst1, sst2A, sst2B, and sst3 using peptides corresponding to their COOH-terminal sequences. These antisera were used for immunocytochemical staining of paraffin sections of 33 primary breast cancers. Somatostatin receptor-like immunoreactivity (Li) was predominantly localized to the plasma membrane of the tumor cells. In the vast majority of positively stained tumors, somatostatin receptor-Li was uniformly present on nearly all tumor cells. Both the level and the pattern of expression of ssts varied greatly between individual carcinomas. sst2A-Li and/or sst2B-Li was detectable in 28 tumors (85%); among these, 14 tumors (42%) showed particularly high levels of sst2-Li. sst1-Li was found in 17 (52%) cases and sst3-Li in 16 (48%) cases. The expression of ssts was independent of patient age, menopausal status, **diagnosis**, histological grade, and levels of estrogen and progesterone receptors. The immunocytochemical determination of somatostatin receptor status allows direct detection of receptor protein on the tumor cells and, hence, may provide more precise information than reverse transcription-PCR for predicting response to octreotide therapy in breast cancer.

L8 ANSWER 8 OF 141 MEDLINE
AN 1998248312 MEDLINE DUPLICATE 3
DN 98248312
TI Growth characteristics and metastatic properties of human breast cancer xenografts in immunodeficient mice.
AU Visonneau S; Cesano A; Torosian M H; Miller E J; Santoli D
CS The Wistar Institute of Anatomy and Biology, Philadelphia, Pennsylvania 19104, USA.
SO AMERICAN JOURNAL OF PATHOLOGY, (1998 May) 152 (5) 1299-311. Journal code: 3RS. ISSN: 0002-9440.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199808
EW 19980801
AB We evaluated the growth and metastatic potential of two human **breast cancer cell lines** and 16 patient-derived biopsy specimens, representing the most common histological types of breast carcinomas, upon subcutaneous implantation into severe combined immunodeficient (SCID) mice. The method of engraftment we used, based on implantation of intact tissue specimens and complete immunosuppression of the host, provided an easier system to grow human breast carcinoma specimens in mouse models and resulted in a 50% success rate of tumor take. No correlation was found between growth in SCID mice and pathological **diagnosis**, grading, or estrogen/progesterone receptor expression by the tumor biopsy specimen. Serial passage of the tumor fragments in SCID mice resulted in increased metastasis rates and more rapid emergence of a palpable tumor mass. A tumor from a patient with infiltrating ductal carcinoma, which grew aggressively and metastasized in 100% of the female SCID mice, was also successfully engrafted in 100% of nonobese diabetic (NOD)/SCID female mice, but systemic spread was minimal. Fragments of the same tumor grew in

only 33% of male SCID mice with very limited metastases. A strong correlation (r = 0.997) was observed between tumor burden and the presence of soluble (serum) interleukin-2 receptor, a marker associated with a subset of human breast tumors. All together, these data indicate the usefulness of SCID/human breast tumor xenografts for measuring tumor progression and evaluating novel therapeutic approaches to breast cancer.

L8 ANSWER 9 OF 141 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:512747 BIOSIS
DN PREV199800512747
TI Specificity of reverse transcriptase polymerase chain reaction assays designed for the detection of circulating cancer cells is influenced by cytokines in vivo and in vitro.
AU Jung, R.; Krueger, W. (1); Hosch, S.; Holweg, M.; Kroeger, N.; Gutensohn, K.; Wagener, C.; Neumaier, M.; Zander, A. R.
CS (1) Bone Marrow Transplantation Centre, Dep. Oncol./Haematol., Univ. Hosp. Eppendorf, Martinistrasse 52, 20246 Hamburg Germany
SO British Journal of Cancer, (Nov., 1998) Vol. 78, No. 9, pp. 1194-1198. ISSN: 0007-0920.
DT Article
LA English
AB Several reverse transcriptase polymerase chain reaction (RT-PCR) assays have been described for the detection of circulating tumour cells in blood and bone marrow. Target mRNA sequences for this purpose are the cytokeratins (CK) 19 and 20, the carcinoembryonic antigen (CEA), and the prostate-specific antigen messages. In this study, we investigated biological factors influencing the specificity of the CK19 and CEA RT-PCR assays. Bone marrow, granulocyte colony-stimulating factor (G-CSF)-mobilized blood stem cells and peripheral blood samples obtained from healthy volunteers (n = 15; CEA n = 7), from patients with epithelial (n = 29) and haematological (n = 23) cancer and from patients with chronic inflammatory diseases (n = 16) were examined. Neither CEA nor cytokeratin 19 messages could be amplified from bone marrow samples from healthy subjects and from patients with haematological malignancies. In contrast, specimens from patients with inflammatory diseases scored positive up to 60%. To investigate the influence of inflammation on target mRNA expression, haemopoietic cells were cultured with and without cytokine stimulation in vitro. CK19 messages could be easily detected in cultured marrow cells without further stimulation, CEA messages only after gamma-interferon (rINF) stimulation. In contrast, G-CSF-mobilized peripheral blood stem cells were positive for CK19 messages only after stem cell factor (SCF) or interleukin stimulation. We conclude that transcription of so-called tissue-specific genes is inducible in haemopoietic tissues under certain conditions. These factors have to be considered in future applications of RT-PCR for the detection of minimal residual disease.

L8 ANSWER 10 OF 141 MEDLINE
AN 1998272822 MEDLINE
DN 98272822
TI Untreated primary lung and breast cancers: correlation between F-18 FDG kinetic rate constants and findings of in vitro studies.
AU Torizuka T; Zasadny K R; Recker B; Wahl R L
CS Department of Internal Medicine, University of Michigan Medical Center, Ann Arbor 48109-0028, USA.

NC CA52880 (NCI)
CA53172 (NCI)
CA56731 (NCI)
SO RADIOLOGY, (1998 Jun) 207 (3) 767-74.
Journal code: QSH. ISSN: 0033-8419.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199808
EW 19980803
AB PURPOSE: To compare kinetic modeling of 2-[fluorine-18]fluoro-2-deoxy-D-glucose (F-18 FDG) between untreated primary lung and untreated primary breast cancers by using positron emission tomographic (PET) findings and to correlate these findings with findings of in vitro studies. MATERIALS AND METHODS: Nineteen patients (12 men, seven women; age range, 49-82 years) with untreated primary lung cancer and 17 women with untreated primary breast cancer (age range, 26-65 years) underwent 1-hour dynamic F-18 FDG PET. A three-compartment model was applied to F-18 FDG kinetics in tumors. The standard uptake value normalized for lean body mass (SUVlean) in tumors was measured 50-60 minutes after tracer injection. In vitro, thin-layer chromatography was performed to evaluate the intracellular phosphorylation of tritiated F-18 FDG in human lung cancer and **breast cancer cell lines**.
RESULTS: At PET, lung cancer had a significantly ($P < .003$) higher rate constant for F-18 FDG phosphorylation (k3) and SUVlean than did breast cancer (0.164 +/- 0.150 [standard deviation] vs 0.043 +/- 0.018 and 8.25 +/- 3.28 vs 3.17 +/- 1.08, respectively). Breast cancer showed a significant correlation between k3 and SUVlean ($r = .607$, $P < .01$), although no such correlation was observed in lung cancer. In vitro studies
showed phosphorylation of F-18 FDG in breast cancer cells was less complete in hyperglycemia than it was in lung cancer cells. CONCLUSION: A much lower k3 appears to be a rate-limiting factor for F-18 FDG accumulation in breast cancer, while the higher k3 in lung cancer is probably not rate limiting for F-18 FDG accumulation.

=> s colon cancer cell line and diagnos?

2 FILES SEARCHED...
L9 85 COLON CANCER CELL LINE AND DIAGNOS?

=> s l9 range=,1998

L10 67 L9

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DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS; CAPLUS'
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L11 ANSWER 1 OF 54 CAPLUS COPYRIGHT 2000 ACS
AN 1998:766513 CAPLUS

DN 130:24100
TI Immunogenic compositions to the CCK-B/gastrin-receptor and methods for the treatment of tumors
IN Michaeli, Dov; Caplin, Martyn; Watson, Susan A.; Grimes, Stephen
PA Aphton Corporation, USA
SO PCT Int. Appl., 60 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9851337	A2	19981119	WO 1998-US9957	19980512
	WO 9851337	A3	19990204		

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CN, CU, CZ, EE, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, RO, RU, SD, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

	AU 9875740	A1	19981208	AU 1998-75740	19980512
	EP 981369	A2	20000301	EP 1998-923447	19980512

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

	BR 9811264	A	20000718	BR 1998-11264	19980512
PRAI	US 1997-46201	19970512			
	WO 1998-US9957	19980512			

AB The invention concerns immunogens, immunogenic compns. and method for the treatment of gastrin-dependent tumors. The immunogens comprise a peptide from the CCK-B/gastrin-receptor conjugated to a spacer and to an immunogenic carrier. The immunogens are capable of inducing antibodies in vivo which bind to the CCK-B/gastrin-receptor in tumor cells, thereby preventing growth stimulating peptide hormones from binding to the receptors, and inhibiting tumor cell growth. The immunogens also comprise antibodies against the CCK-B/gastrin-receptor for passive immunization. The invention also concerns diagnostic methods for detecting gastrin-dependent tumors in vivo or from a tissue biopsy using the antibodies of the invention. CCK-B/gastrin receptor peptides were synthesized, conjugated with diphtheria toxoid, and used as immunogens to raise antibodies in rabbits and to inhibit growth of pancreatic adenocarcinoma and colon cancer cell lines.

L11 ANSWER 2 OF 54 CAPLUS COPYRIGHT 2000 ACS
AN 1998:698780 CAPLUS
DN 130:104887
TI Effect of the combined treatment with 5-fluorouracil, .gamma.-interferon or folinic acid on carcinoembryonic antigen expression in colon cancer cells
AU Aquino, Angelo; Prete, Salvatore P.; Greiner, John W.; Giuliani, Anna; Graziani, Grazia; Turriziani, Mario; De Filippi, Rosaria; Masci, Giovanna; Bonmassar, Enzo; De Vecchis, Liana
CS Department of Neuroscience, Section of Pharmacology and Medical Oncology University of Rome, Rome, 00133, Italy

SO Clin. Cancer Res. (1998), 4(10), 2473-2481
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal
LA English
AB 5-Fluorouracil (5-FU) and human recombinant .gamma.-interferon (.gamma.-IFN) were found to increase the expression of carcinoembryonic antigen (CEA) in human cancer cells in vitro. In the present study, the antimetabolite assocd. with .gamma.-IFN or folinic acid (FA), a biochem. modulator of cellular metab. of 5-FU, had increased antineoplastic activity. Treatment of two human **colon cancer cell lines** (HT-29 and WiDr) with 5-FU + .gamma.-IFN resulted in an increase of CEA expression higher than that obtainable with both agents alone, although no synergistic effects were obtained. This was demonstrated in terms of: (a) mRNA transcripts (HT-29); (b) cytoplasm and membrane CEA protein levels detected by Western blot anal. (HT-29); and (c) plasma membrane reactivity detd. by flow cytometry anal. (HT-29 and WiDr). Moreover, 5-FU + .gamma.-IFN increased HLA class I mols. in the HT-29 cell membrane over that obtainable with .gamma.-IFN alone. In contrast, both agents did not induce the expression of the costimulatory mol. B7-1. Treatment with FA enhanced the antitumor effect of 5-FU but not its ability to augment CEA expression. This suggests that the FA-sensitive biochem. mechanism of action of 5-FU is not involved in its effect on CEA expression. In vivo studies showed, for the first time, that 5-FU, alone or combined with .gamma.-IFN, increases the amt. of CEA protein over controls, either in cancer cells or in peripheral blood of nude mice bearing HT-29 cells. These results could be of potential **diagnostic** and/or therapeutic value when CEA protein is the target of humoral or cell-mediated immunity.
RE.CNT 50
RE
(2) Boyer, C; Cancer Res 1989, V49, P2928 CAPLUS
(3) Chen, C; Cancer Res 1995, V55, P3873 CAPLUS
(4) Cheng, Y; J Biol Chem 1985, V260, P15834 CAPLUS
(5) Chomczynski, P; Anal Biochem 1987, V162, P156 CAPLUS
(6) Chu, E; Cancer Res 1990, V50, P5834 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 54 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:512747 BIOSIS
DN PREV199800512747
TI Specificity of reverse transcriptase polymerase chain reaction assays designed for the detection of circulating cancer cells is influenced by cytokines in vivo and in vitro.
AU Jung, R.; Krueger, W. (1); Hosch, S.; Holweg, M.; Kroeger, N.; Gutensohn, K.; Wagener, C.; Neumaier, M.; Zander, A. R.
CS (1) Bone Marrow Transplantation Centre, Dep. Oncol./Haematol., Univ. Hosp. Eppendorf, Martinistrasse 52, 20246 Hamburg Germany
SO British Journal of Cancer, (Nov., 1998) Vol. 78, No. 9, pp. 1194-1198. ISSN: 0007-0920.
DT Article
LA English
AB Several reverse transcriptase polymerase chain reaction (RT-PCR) assays have been described for the detection of circulating tumour cells in blood and bone marrow. Target mRNA sequences for this purpose are the cytokeratins (CK) 19 and 20, the carcinoembryonic antigen (CEA), and the prostate-specific antigen messages. In this study, we investigated

biological factors influencing the specificity of the CK19 and CEA RT-PCR assays. Bone marrow, granulocyte colony-stimulating factor (G-CSF)-mobilized blood stem cells and peripheral blood samples obtained from healthy volunteers (n = 15; CEA n = 7), from patients with epithelial (n = 29) and haematological (n = 23) cancer and from patients with chronic inflammatory diseases (n = 16) were examined. Neither CEA nor cytokeratin 19 messages could be amplified from bone marrow samples from healthy subjects and from patients with haematological malignancies. In contrast, specimens from patients with inflammatory diseases scored positive up to 60%. To investigate the influence of inflammation on target mRNA expression, haemopoietic cells were cultured with and without cytokine stimulation in vitro. CK19 messages could be easily detected in cultured marrow cells without further stimulation, CEA messages only after gamma-interferon (rINF) stimulation. In contrast, G-CSF-mobilized peripheral blood stem cells were positive for CK19 messages only after stem cell factor (SCF) or interleukin stimulation. We conclude that transcription of so-called tissue-specific genes is inducible in haemopoietic tissues under certain conditions. These factors have to be considered in future applications of RT-PCR for the detection of minimal residual disease.

L11 ANSWER 4 OF 54 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:450695 BIOSIS
DN PREV199800450695
TI Signal transduction mechanisms in neurotensin-mediated cellular regulation.
AU Ehlers, Richard A., II; Bonnor, Ricardo M.; Wang, Xiaofu; Hellmich, Mark R.; Evers, B. Mark (1)
CS (1) Dep. Surg., Univ. Tex. Med. Branch, 301 University Blvd., Galveston, TX 77555-0533 USA
SO Surgery (St Louis), (Aug., 1998) Vol. 124, No. 2, pp. 239-247. ISSN: 0039-6060.
DT Article
LA English
AB Background. Neurotensin, an important gut hormone, binds its receptor (NTR) to stimulate proliferation of intestinal cells; the molecular mechanisms remain largely undefined. Mitogen-activated protein kinases (MAPKs) translocate to the nucleus and induce transcription factors (e.g., c-Fos) in response to certain trophic agents. The purpose of this study was (1) to define the signaling mechanisms regulating neurotensin and (2) to determine whether the AP-1 transcription factor c-Fos is induced. Methods. Expression of the NTR gene was determined in the human **colon cancer cell lines** KM12C, KML4A, and KH20 by Northern blot analysis and ribonuclease-protection experiments. To assess whether NTR was functionally coupled to the Ca2+-signaling pathway, intracellular Ca2+ ((Ca2+)i) mobilization was assessed by fura-2 spectrofluorometry. To determine whether the MAPK signaling pathway was activated in KM20 cells by neurotensin, Western blots for the phosphorylated forms of MAPK (p42 and p44) and in vitro kinase assays were performed. In addition, Western blots were performed to assess levels of c-Fos after neurotensin treatment. Results. The NTR gene was expressed in the KM20 cell line but not in KA12C or KM12LA. The NTR in KM20 cells was functionally coupled to the Ca2+-signaling pathway as noted by a rapid (30 seconds) mobilization of (Ca2+)i after addition of

neurotensin; the neurotensin; the neuotensin-mediated response was block by the NTR antagonist SR48692. Both p42MAPK and p44MAPK were stimulated by neurotensin apprx3 to 6 minutes after treatment. Increased levels of c-Fos were demonstrated, with peak levels occurring 2 hours after addition of neurotensin. Conclusions. Our results demonstrate that neurotensin binds to its endogenous NTR in KM20 cells with stimulation of the Ca2+- and MAPK-signaling pathways and an increase in the levels of the AP-1 protein c-Fos. Delineating the signal transduction mechanisms regulating the cellular effects of neurotensin will provide important insights into the molecular pathways responsible for gut hormone-mediated proliferation.

L11 ANSWER 5 OF 54 MEDLINE
AN 1998061047 MEDLINE
DN 98061047
TI Differential effects of deoxycholic acid on proliferation of neoplastic and differentiated colonocytes in vitro.
AU Peiffer L P; Peters D J; McGarrity T J
CS Department of Medicine, University Hospital, Milton S. Hershey Medical Center, Pennsylvania State University, Hershey 17033, USA.
NC R29 CA45468 (NCI)
SO DIGESTIVE DISEASES AND SCIENCES, (1997 Nov) 42 (11) 2234-40.
Journal code: EAD. ISSN: 0163-2116.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199803
EW 19980302
AB The secondary bile acid deoxycholic acid is believed to be a promoter of large bowel cancer, in part by inducing colonic epithelial proliferation. The effects of deoxycholic acid on [3H]thymidine incorporation by the human **colon cancer cell** line HT29 and two differentiated subclones were measured and compared. The subclone HT29-C1 has features of mature absorptive cells and HT29-N2 cells secrete mucus under cholinergic control. The three cell lines were treated with deoxycholic acid (DCA) at concentrations of 0, 5, 10, 50, 100, 150, and 300 microM for 3, 6, 9, 15, 24, and 48 hr. A significant increase in proliferation was noted in HT29 cells only at 6 hr with 5 and 10 microM deoxycholic acid. Neither the subclone HT29-C1, nor HT29-N2 cells exhibited significant change in [3H]thymidine incorporation with DCA at these concentrations or time points. Higher doses of deoxycholic acid above 50 microM and duration of exposure greater than 24 hr were cytotoxic to all three cell lines. The proliferative effects of DCA in HT29 cells were not paralleled by changes in protein kinase C activity or protein kinase C isoform expression. Quantitative and qualitative differences in PKC isoform expression were not noted in the three cell lines used in this study. The proliferative effects of DCA on HT29 cells appear to be independent of the PKC signal transduction pathway.

L11 ANSWER 6 OF 54 MEDLINE DUPLICATE 1
AN 1998021373 MEDLINE
DN 98021373
TI Expression of alpha1-6 fucosyltransferase in rat tissues and human cancer cell lines.
AU Miyoshi E; Uozumi N; Noda K; Hayashi N; Hori M; Taniguchi N
CS Department of Biochemistry, Osaka University Medical School, Suita, Japan.

SO INTERNATIONAL JOURNAL OF CANCER, (1997 Sep 17) 72 (6) 1117-21.
Journal code: GQU. ISSN: 0020-7136.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Cancer Journals; Priority Journals
EM 199801
AB GDP-L-Fuc:N-acetyl-beta-D-glucosaminide alpha1-6 fucosyltransferase (alpha1-6FucT) catalyzes the transfer of a fucosyl residue from GDP-fucose to the asparagine-linked GlcNAc residue of complex N-glycans via alpha1-6 linkage. These oligosaccharide structures are essential for the attachment of polysialic acid to the neural-cell-adhesion molecule, and its levels are useful for the differential **diagnosis** of hepatocellular carcinomas with respect to the microheterogeneity of alpha-fetoprotein. We have been successful in the purification and cDNA cloning of alpha1-6FucT from porcine brain and from a human gastric-cancer cell line. In the present study, mRNA expression of alpha1-6FucT in various rat tissues and human cancer cell lines was examined, along with the expression of alpha1-6FucT mRNA and the induction by treatment with several cytokines. Northern-blot analysis indicated high expression levels of alpha1-6FucT in brain and gastrointestinal-tract tissues of normal rats, as well as for a number of lung-cancer, gastric-cancer and **colon-cancer cell lines**. Although various cytokines did not induce alpha1-6FucT mRNA, differentiation of a tumor cell enhanced the mRNA by 2- to 3-fold. These results may provide new insight into studies on alpha1-6FucT in terms of carcinogenesis or differentiation.

L11 ANSWER 7 OF 54 MEDLINE
AN 97364905 MEDLINE
DN 97364905
TI Possible effect of pneumoperitoneum on the spreading of colon cancer tumor cells.
AU Chen W S; Lin W; Kou Y R; Kuo H S; Hsu H; Yang W K
CS Division of Colorectal Surgery, Veterans General Hospital-Taipei, College of Medicine, National Yang-Ming University, Taiwan.
SO DISEASES OF THE COLON AND RECTUM, (1997 Jul) 40 (7) 791-7.
Journal code: EAB. ISSN: 0012-3706.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199710
EW 19971002
AB PURPOSE: By using a murine hepatic metastatic model, we tried to investigate the possible influence of gas insufflation in colon cancer cells spreading from the portal system to the liver. METHODS: After transducing the human placental ALP gene into murine **colon cancer cell line** CT26, we successfully selected a clone of CT26/DAP that would yield a specific color following histochemical staining. Fifty mice were assigned into two groups, receiving either an intrasplenic injection of 10(6) CT26/DAP cells alone or the cells followed by intra-abdominal helium insufflation with the pressure of 15 cm H2O for ten minutes. Five mice in each group were used to observe their survival and the other mice were killed at four different

time periods: 10 minutes, 24 hours, 48 hours, and 72 hours following cell injection. The livers and spleens were removed for histochemical staining.
By counting the numbers of specific dark reddish spots of CT26/DAP cells, we could estimate the number of tumor cells on the hepatic surface. RESULTS: At the very beginning following tumor cell injection, we found a significantly greater number of tumor cells on the hepatic surface in mice with gas insufflation (6354 +/- 1072 vs. 2133 +/- 223, respectively; P = 0.012). But the difference of these two groups became smaller and smaller as time went by. The number of tumor cells on the hepatic surface would reach the lowest level at postoperative 48 hours, and the tumor foci then began to grow both in size and number. The above patterns of dynamic change in tumor cell distribution were similar in mice both with and without gas insufflation. Average survival was slightly shorter in mice with gas insufflation, but the difference was not statistically significant. CONCLUSION: Pneumoperitoneum caused by gas insufflation may increase tumor cell spread from the portal system to the liver at the very beginning stage; however, there was no significant difference in long-term survival between mice with and without gas insufflation in this murine animal model.

L11 ANSWER 8 OF 54 CAPLUS COPYRIGHT 2000 ACS
AN 1997:172112 CAPLUS
DN 126:304447
TI Preparation of antibodies against hMSH2 and hMLH1 and its cellular localization in human **colon cancer cell lines**
AU Kikuchi, Yoshinori; Takano, Shoichi
CS Sch. Med., Toho Univ., Tokyo, 143, Japan
SO Toho Igakkai Zasshi (1997), 43(5), 432-445
CODEN: TOIZAG; ISSN: 0040-8670
PB Toho Daigaku Igakkai
DT Journal
LA English
AB Proteins responsible for DNA mismatch repair (MMR) system are assocd. with post-replication mismatch repair and transcription-coupled repair (TCR). To study expression of MMR genes at the protein level, the authors prepd. the antibodies against genetically engineered hMSH2 and hMLH1 proteins. The expression plasmids, pMS56 which encodes codons 603-900 of the hMSH2 gene and pML56 which encodes codons 521-752 of the hMLH1 gene were constructed and induced to express the proteins. Recombinant hMSH2 (rhMSH2) and hMLH1 (rhMLH1) proteins were purified and used for immunization. By immunoblot anal., anti-hMSH2 monoclonal antibodies SFA3 and SAD4 recognized 100 kDa protein in the hMSH2-proficient **colon cancer cell lines** SW-620 and HCT 116. The anti-hMLH1 monoclonal antibody LEC12 recognized 80 kDa protein in the hMLH1-proficient cell lines SW-620 and LoVo. Nucleus of all SW-620 cells, esp. mitotic cells, were stained with these antibodies. These results indicate that both hMSH2 and hMLH1 proteins may be present at any phase of the cell cycle and localized in the cellular nucleus. These results support that MMR gene products assoc. not only with post-replication mismatch repair, which is expected to occur in the S phase of the cell cycle, but also with TCR, which may be independent of the cell cycle. These antibodies may be useful for detection of MMR gene products in clin.

specimens and helpful for reaching a **diagnosis** of certain cancers.

L11 ANSWER 9 OF 54 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:232775 BIOSIS
DN PREV199799531978
TI Bifunctional ligand attachment to P-glycoprotein specific antibody, UIC2: Applications to tumor imaging and radioimmunotherapy.
AU Thakar, J. H.; Okunieff, P.; Brechbiel, M.; Garmestani, K.; Robey, R.; Bates, S. E.; Carrasquillo, J. A.; Abraham, E. H.
CS Radiation Oncol. Branch, NCI, Bethesda, MD 20892 USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1997) Vol. 38, No. 0, pp. 384.
Meeting Info.: Eighty-eighth Annual Meeting of the American Association for Cancer Research San Diego, California, USA April 12-16, 1997
ISSN: 0197-016X.
DT Conference; Abstract
LA English

L11 ANSWER 10 OF 54 CAPLUS COPYRIGHT 2000 ACS
AN 1996:625609 CAPLUS
DN 125:245707
TI Monoclonal antibodies specific for human matrix metalloproteinase 7 (MMP-7)
IN Okada, Yasunori; Oochi, Eiko; Yamazaki, Tomomi; Tono, Isao; Yoshida, Shinichi; Iwata, Kazushi
PA Fuji Yakuhin Kogyo Kk, Japan
SO Jpn. Kokai Tokkyo Koho, 18 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 08217800	A2	19960827	JP 1995-50375	19950216
	JP 2949467	B2	19990913		

AB Disclosed are monoclonal anti-human MMP7 antibodies and sandwich immunoassay using the antibodies for quantification of human MMP7. These antibodies are specific for N-terminal amino acid sequence of human MMP-7, human pro-MMP-7, C-terminal of human MMP-7, intermediate of human MMP-7, or active human MMP-7. The monoclonal antibodies are esp. useful for **diagnosis** of colorectal cancer, prostate cancer, or other cancers. In example, pro-MMP-7 derived from human **colon cancer cell line** CaR-1 was purified, polypeptides of the pro-MMP-7 were prepd. and used to raise monoclonal antibodies, these antibodies were modified and used for immunostaining **diagnosis** of stomach cancer or quantitation of human MMP-7 in blood serum.

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